Fish Disease Leaflet 84

Bacterial Gill Disease of Freshwater Fishes[1]

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Introduction

Fish gills function as both respiratory and excretory organs. Basically they consist of a network of capillaries where blood is separated from the surrounding water by only one or two layers of cells. Proliferation of epithelial tissue, and later the loss of surface by the clubbing and fusing of lamellae, impair respiration and the excretion of nitrogenous waste materials, and disturb osmotic balance. Because these changes adversely affect the health of fish, the prevention and treatment of gill diseases are important in fish culture.

Pathologic changes in gill tissues have been divided into five categories: (1) bacterial gill disease (BGD) of salmonids and pondfishes, caused by filamentous bacteria that have yellow pigment in their cell walls; (2) nutritional gill disease, caused by a deficiency of pantothenic acid; (3) hemorrhagic gill disease, in which an apparent hemorrhagic condition is really a dilation of lamellar vessels (telangiectasia), caused by toxic agents or physical injury; (4) mycotic gill necrosis in pondfishes, caused by the fungus *Branchiomyces* (Meyer and Robinson 1973; Niesh and Hughes 1980); and (5) proliferative gill disease of unknown etiology in pondfishes and rainbow trout, *Oncorhynchus mykiss* (Daoust and Ferguson 1985). Bacterial gill disease, the most lethal of these categories, is discussed here.

Etiology

Outbreaks of BGD are triggered by stressors that result from intensive culture. Although crowding, low dissolved oxygen concentrations, and ammonia buildup may contribute to epizootics, the exact role of these and other stressors is not known. However, when proper stressors are present, gill tissues are colonized by filamentous yellow-pigmented bacteria that damage gill tissue and cause death of the fish. Davis (1927) first observed the bacteria and gill damage in trout at a Vermont hatchery. Later, Rucker et al. (1949, 1952) and Bullock (1972) isolated several strains of yellow-pigmented bacteria from infected gill tissue but none could be shown to be the causative agent. Wakabayashi (1980) isolated a different yellow-pigmented bacterium and experimentally produced BGD with this organism. The bacterium, named *Flavobacterium branchiophila* by Wakabayashi et al. (1989), is probably the cause of most BGD outbreaks in salmonids.

Gill lesions in warmwater pond fish are produced by *Cytophaga* (formerly *Flexibacter*) *columnaris*, the causative agent of columnaris disease. Although columnaris disease is typically characterized by fins and body lesions, gill pathology involving this bacterium has been reported (Foscarini 1989; Farkas and Olah 1986).

Pathology

In disease caused by *F. branchiophila*, a proliferative hyperplasia develops in the epithelium of the gill lamellae. In a typical case, electron microscopic studies of affected gills show hypertrophy of lamellar epithelium in the early stages of the disease, which results in a transfiguration of flat epithelial cells into cuboidal or columnar cells and oval chloride cells. As the disease progresses, the epithelium proliferates, causing clubbing and fusing of gill lamellae. Also, staining of gill tissue with ruthenium red reveals bacterial cells adhering to the outermost epithelial layer (Kudo and Kimura 1983a). The adhesion of *F. branchiophila* to gill tissue may be significant because Kudo and Kimura (1983b) demonstrated that an extract from *F. branchiophila* produced the pathological changes seen in a natural outbreak. Later, the same authors (Kudo and Kimura 1983c) found that, after treatment, 5 weeks were required for complete recovery of gill tissue.

Necrosis of gill tissue rather than hyperplasia occurs in BGD infections caused by *C. columnaris*. Farkas and Olah (1986) described three stages of *C. columnaris* infection of gills (termed gill necrosis) in common carp (*Cyprinus carpio*). The first stage is characterized by gills that are either pale or dark purple, caused by stressors such as ammonia, unfavorable temperatures, or toxins. No *C. columnaris* cells can be seen in stage 1 but are present in damaged gills in stage 2 and cause necrosis and mortality. In stage 3 (if fish survive), *C. columnaris* disappears but the necrotic area does not become functional. Funahashi (1980) reported different pathological changes in gill infections of the European eel (*Anguilla anguilla*) and Japanese eel (*A. japonicus*). In Japanese eels, there was hyperplasia of gill epithelium, and in advanced cases, filaments became necrotic and were sloughed off. In European eels, the clubbing of filaments was caused by hyperplasia of mucous cells but necrosis was not seen. Foscarini (1989) found that pathological changes in gill tissue of eels caused by *C. columnaris* infections resulted in alterations in cardiac performance. Bradycardia occurred when gills were hyperplastic, and a compensatory tachycardia developed as gill tissue became necrotized. He hypothesized that a combination of impaired gill circulation and cardiac performance results in the death of fish.

Diagnosis

Preliminary diagnosis of BGD in salmonids is based on the behavior of affected fish: they usually stop feeding,

swim at the surface, and often orient themselves upstream (against the current) to ensure efficient water flow over gill surfaces. Because these behavior patterns usually do not occur in pondfishes with BGD, the disease cannot be diagnosed until mortalities begin. Definitive diagnosis is based on the examination of wet mounts of gill tissue for hyperplasia or necrosis and the presence of long, thin gram-negative bacteria. The causative agent is identified as *F. branchiophila* or *C. columnaris* by isolation and application of definitive biochemical tests. A fluorescent antibody test has been developed for *F. branchiophila* (Huh and Wakabayashi 1987).

Host and Geographic Range

Bacterial gill disease has been reported from a broad range of cultured coldwater and warmwater fishes. All cultured species are probably susceptible because disease outbreaks are closely associated with stress caused by intensive culture. Although the original description of BGD was made in North America, the condition occurs worldwide.

Source and Reservoir of Infection

The source of *F. branchiophila* is not known. The bacterium may be a normal inhabitant of the water or gills. Although *C. columnaris* can live in water and mud, fish probably serve as the main reservoir of infection.

Incubation and Communicability

The incubation period for either *F. branchiophila* or *C. columnaris* is variable because disease outbreaks depend on the presence of stressors. Bullock (1972) induced BGD in fingerling rainbow trout in 10 to 14 days when the fish were subjected to crowding, dissolved oxygen concentrations of 4 to 5 ppm, and ammonia concentrations of 1 ppm.

Control

Prevention

Prevention of BGD in hatchery salmonids is difficult because the fish are constantly stressed. Sanitation and application of the proper cultural practices is helpful, but does not eliminate outbreaks. To help reduce such outbreaks, many fish culturists use prophylactic chemotherapy with chemicals (described later) once or twice a month.

Prevention of BGD is more difficult in pondfishes than in hatchery fishes, because *C. columnaris* usually occurs in the water source.

Treatment

Several chemicals have been used to treat BGD in hatchery salmonids. The most widely used are quaternary ammonium compounds, such as benzalkonium chlorides, available as Hyamine 1622 (98.8% active ingredient) and Hyamine 3500 (50% active ingredient). Another compound of this type is Roccal, available in 10% or 50% concentrations. Benzalkonium chlorides are used in concentrations of 1 to 2 ppm (calculated on the basis of the active ingredient) as a 1-h bath or continuous flow treatment. Caution is necessary because the margin of safety is narrow, particularly in soft water.

Another chemical is the herbicide Diquat. It has been used at a concentration of 8.4 to 16.8 ppm of the formulated material, or 2 to 4 ppm on the basis of active ingredient (Diquat cation).

None of these chemicals are approved by the U.S. Food and Drug Administration for disease control in food fishes. Efforts are under way to have chloramine-T registered as a treatment for BGD. This compound was found effective for BGD when used at 8.5 ppm for 1 h (From 1980). In hatchery tests conducted by the National Fish Health Research Laboratory, one treatment effectively controlled BGD when it was given early in an outbreak. However, two or more treatments are required for advanced outbreaks.

When BGD, caused by *C. columnaris*, occurs in pondfish, external treatments for the control of columnaris disease are used. The treatment with 8.5 ppm Diquat described above has been reported to be effective. Diquat, copper sulfate at 0.5 ppm, and potassium permanganate at 2 to 4 ppm can be added to ponds and allowed to dissipate over time. If copper sulfate or potassium permanganate are used, treatment levels may have to be adjusted, depending on water chemistry. In soft water, 0.5 ppm copper sulfate may be toxic; and if pondwater is high in organic material, potassium permanganate concentrations must be increased (Rogers 1971; Jee and Plumb 1981).

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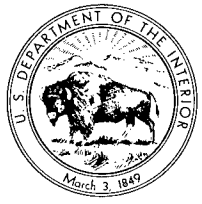

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